Cigarette smoke on coronary vascular resistance. The interpretation of the perfusion experiments by (8) Aviado et al and (38) Folle et al is not complete. The authors mention a second explanation in that the amount of nicotine absorbed in the lungs is not sufficient to influence the coronary vessels. The quotation from Folle et al is as follows:

> The perfused coronary artery behaved in the following manner: no immediate change when blood exposed to cigarette smoke was perfused into the coronary artery but an increase in coronary vascular resistance when nicotine was injected directly into the vessel. There are two possible explanations for this discrepancy: (*a*) that the amount of nicotine absorbed from the left lower lobe was less than the 1μg/kg injected directly into the perfused artery; or (*b*) that blood exposed to cigarette smoke did not act in the same manner as pure nicotine. In regard to the latter possibility, nicotine may be in the blood in sufficient quantities but there was an additional amount of substance released which antagonizes the primary actions of nicotine. Such a substance may be histamine which is known to reduce coronary vascular resistance,[23] and this may serve to reduce any effect of nicotine. These possibilities can be verified by determination of nicotine in the blood. Until a sensitive method becomes available, it is important to question the exclusive use of nicotine in the understanding of the coronary effects of tobacco.
>
> The systemic absorption of substances from the lungs exposed to cigarette smoke initiated stimulation of the sympathetic nervous system with an elevation in aortic blood pressure and positive inotropic action on the heart muscle. The latter was accompanied by an elevation in coronary vascular resistance, although the perfusing blood was not exposed to cigarette smoke. Both the changes in coronary vascular resistance and myocardial contractility are indications of generalized increase of sympathetic nervous activity. If the coronary artery was not perfused, the coronary vasomotor effect would have been masked by an actual elevation in aortic pressure and the entrance of catecholamines released from the adrenal medulla so that the end result would have been an actual increase in coronary blood flow. This conforms to the general conclusion in an earlier paper[24] that the cardiac effects of tobacco arise almost entirely from the extracardiac actions of smoking instead of a direct response of the heart.

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